ON THE

DISAPPEARANCE OF THE AORTIC REGURGITANT MURMUR.

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The mutability of murmurs originating at the mitral and tricuspid orifices has been for a long time well known, and was excellently illustrated a good many years ago by the late Professor W. R. Sanders in this Journal.[1] In the same paper he expressed the opinion that murmurs originating at the aortic orifice do not undergo similar variations, but are always to be heard by a well-trained auscultator. I had always fully accepted this doctrine, subject only to a faint remembrance of a protest emanating from Professor Gairdner of Glasgow; and though I, in common, I suppose, with other physicians who follow their cases to the post-mortem table, have had sometimes to hear aortic valves condemned as incompetent over which during life I had failed to detect a murmur, I did not venture to formulate a doubt of its soundness until a recent experience supplied me with evidence which I could not hesitate to accept.

On 8th February 1885, Dr Alfred Harvey brought to consult me at my house Mr W., aged 48, who complained of sharp shooting pains in his legs and thighs, dimness of sight, slight palpitation, loss of appetite, and indigestion. There was no headache or giddiness. The patellar reflexes were present. A diastolic murmur was audible in the aortic area, propagated down the sternum. The urine was free from albumen and sugar.

In the summer of 1885 he began to suffer from dyspnœa and dropsy, and being reduced in circumstances from his long illness, on 9th June 1886—that is, a year after the commencement of the more urgent symptoms of heart disease—he was admitted into the General Hospital under Dr Wade, who kindly transferred him to my care. (Dr Harvey informs me that the murmur had remained audible during the whole of the interval between my first seeing him and some time shortly before his admission to hospital, but he is unable to fix the precise date.) After his admission the

[1] *Edinburgh Medical Journal*, January 1869.

following note was made of the state of the heart:—The apex beat is in the sixth left intercostal space, ¾ inch to the left of the vertical nipple line, while to the right of the sternum the area of dulness is increased. There is a systolic murmur at the apex, followed by an accentuated second sound. At the base the second sound is *reduplicated*, and accentuated in the pulmonary area. There is no aortic diastolic murmur. The radial pulse is regular, small, and the artery hard. There is general anasarca, with effusion into the bases of both pleural sacs, œdema of the lungs, and the urine, which is scanty, contains a trace of albumen, 1·5 per cent. of urea, and deposits granular casts.

I need not go into the details of the progress of the case. His breathing assumed the Cheyne-Stokes type, his mental faculties became obscured, and he died comatose on 11th July, rather more than a month after his admission.

As I had placed a copy of my private notes on the history of the case, it may be supposed that it was a matter of considerable interest to me, and to our resident medical officer Dr Wilson, to try to detect some sign of the lost aortic murmur. In fact, it was listened for repeatedly by myself and by many others, but at no time could it be heard, and I attended the post-mortem examination with more than usual interest. This was performed by our pathologist Dr Crooke, and the following is his account of the state of the heart. It weighed 17 oz., the aorta was dilated, its inner surface atheromatous, and its valves *incompetent*. The left ventricle was enormously hypertrophied, its muscular wall showing patches of *interstitial myocarditis*, with some grayish or yellowish cloudy mottling. The coronary arteries were dilated and atheromatous. The mitral orifice was narrowed, and its valves a little thickened. The right auricle was much distended with blood-clot. The right ventricle was not dilated.

There was no doubt of the incompetence of the aortic valves. I could not but believe that the murmur I had noted was due to this incompetence, and had disappeared during the later period of the disease. Having obtained this proof of the disappearance of an aortic regurgitant murmur, I felt able to place a similar interpretation on the following cases:—

Case II.—J. W. M., aged 30, was admitted on 4th April 1885, complaining of swelling of the legs, cough, and shortness of breath. The cough began two months previously, but the dropsy had existed only nine days before admission. There was a history of two attacks of rheumatic fever. The heart's apex was in the fifth left interspace, diffused and heaving. There was a harsh systolic murmur, loudest at the apex, but traceable round to the angle of the scapula. The second sound was accentuated at the apex and left base, but less loud in the aortic area. There were the physical signs of œdema of the lungs, and the urine was scanty and

albuminous, depositing a few hyaline casts. On the 12th of April, that is, on the ninth day of his residence in the hospital, for the first time a double soft murmur was audible to the right of the sternum, the apical harsh systolic murmur also persisting. He died the same night while trying to get out of bed in a state of delirium.

At the autopsy the pericardium was found to be universally adherent, and the heart in its sac weighed 34 oz. The *aorta* was slightly atheromatous, and its valves *incompetent*. The mitral orifice admitted three fingers, and its valves were thick and stiff. The wall of the *left ventricle was thick, but pale and soft*. The right side of the heart was dilated and hypertrophied, and the tricuspid orifice admitted four fingers.

Case III.—W. R., aged 20, was admitted on 9th February 1885 with general dropsy. He had had ten attacks of rheumatic fever, and the dropsy had been gradually coming on for six months before admission. The heart's apex was in the sixth left interspace in the anterior axillary line. The first sound at the apex was obscure, but there was no distinct murmur. The second sound was loud and clear. In the tricuspid area there was a murmur replacing the first sound. The pulmonary second sound was accentuated. The pulse was small, compressible, and regular. The daily quantity of urine was 30–40 oz. It was slightly albuminous, and deposited a few oxalate of lime crystals and hyaline casts. No other murmurs were ever heard, and the diagnosis attempted was tricuspid and mitral incompetence, with dilatation of the heart, degeneration of the heart's wall, adherent pericardium, and secondary congestion of lungs, liver, spleen, kidneys, etc. He died on 13th February, having been in hospital only five days; and the autopsy was made on the following day. The heart weighed 24 oz., and *the pericardium was attached to it* by old œdematous adhesions. The whole organ was greatly hypertrophied. The aorta was smooth and elastic, but *its valves were thickened and incompetent*. The mitral valve was very much narrowed and slit-shaped, admitting only the tip of the forefinger. The right side of the heart was full of clots. The tricuspid orifice was dilated, but the valves were healthy.

Case IV.—T. S., aged 40, was admitted with dropsy of the lower extremities and tightness of the chest. He had been ill only a month, and the first symptoms were swelling of the face and hands, with scanty urine. There was no history of rheumatism except in the way of vague pains. The apex beat of the heart was in the fifth left interspace. There was no murmur. The aortic second sound was audible, but is noted as being *not* accentuated. His urine was scanty, 20–30 oz. daily, 1025, nearly solid with albumen, containing blood, 1·3 per cent. of urea, and depositing hyaline, epithelial, granular, and blood casts. The case was one of Bright's disease, and he died of cardiac failure on April

7th, ten days after admission. At the post-mortem examination the kidneys were large and mottled, with slightly adherent capsules, but the *heart* weighed 17 oz. The right cavities were stuffed with blood-clot; the tricuspid valve admitted four fingers. The aortic valves were not quite competent, but were adherent, thickened, firm; in parts calcareous. The mitral orifice admitted only the tips of three fingers; its valves were yellow, thickened, and shortened. The wall of the heart showed a moderate degree of *brown atrophy* of its muscular fibres.

The last two cases may be regarded as instances of simple failure to detect a murmur; but if there is no antecedent improbability of the absence of a murmur in spite of the existence of incompetence of the aortic valves, I believe they afford additional illustrations of this fact. Case II. is an instance of the appearance of a murmur while the case was under observation, though it too depends for its probability upon the first case. It is right to state that all the accounts of physical signs are from my own personal observations, having been either noted in my own handwriting or written down to my dictation.

On consulting Professor Gairdner's paper,[1] to which I have to thank him for a reference, I found that his case was that of a man in whom a diastolic murmur made its appearance during his stay in hospital, and disappeared again after a few days. The autopsy showed incompetence of the aortic valves. In a subsequent communication Dr Gairdner repairs an omission by quoting a case mentioned by Walshe,[2] in which a diastolic murmur was replaced by a systolic murmur. These are the only references that I know of in medical literature.

[In the discussion which took place on this paper Dr Lewis Hawkes, house-surgeon at the Children's Hospital, mentioned the disappearance of an aortic regurgitant murmur which had occurred in the wards of Professor T. R. Fraser of Edinburgh while he was acting as clinical clerk. Dr J. A. Lycett, of Wolverhampton, writes to me (9th Dec. 1886) as follows:—"I hoped to get over to Birmingham to-day to hear your paper, in which I should have been interested, having met with several similar instances, and have now under treatment a gentleman with atheromatous arteries, some kidney change, and hypertrophied left ventricle, in whom for several years a loud, diastolic, basic murmur was heard, verified by Sir Walter Foster, in whom now no murmur whatever is to be made out beyond the exaggerated ventricular sound from obstructed circulation through the arteries. He is subject to anginal attacks, and I cannot but look upon his life as very uncertain."]

The explanation accepted by both Walshe and Gairdner is that changes occur in the valve. In Walshe's case he found a mass of vegetations which had blocked the valve, and this certainly

[1] *Brit. Med. Journal*, 1872, vol. i. p. 334.

[2] *Diseases of the Heart*, 3rd edition, 1862.

affords a sufficient explanation. But in none of my cases was there any such evidence, and in Professor Gairdner's case the valves were found post-mortem to be *incompetent*. The explanation I suggest depends upon the general state of the heart, and is strictly analogous to that accepted for the disappearance of mitral murmurs. A buttonhole mitral valve presents physical conditions very similar to those of stiffened and incompetent aortic valves. In each case we might expect, *a priori*, to hear a diastolic murmur; yet when the heart is weak and the circulation feeble we know in the case of the mitral valve that no murmur may be audible. After rest and suitable treatment such a mitral murmur may appear, just as the aortic diastolic murmur did in Case II.

In each of my four cases the heart was *dilated*. In the first there was "interstitial myocarditis," with "grayish or yellowish cloudy mottling;" in the second the wall of the left ventricle was "pale and soft," and the pericardium was "universally adherent;" in the third there was adherent pericardium; and in the fourth there was brown atrophy of the muscular fibres. In every case, moreover, the mitral valve was more or less diseased, so that we had all the conditions present for the propulsion of a very feeble stream of blood into the aorta. Under such circumstances there would be a very feeble aortic recoil, and notably in the first case, but also in some of the others, the coats of this vessel were stiffened and weakened by atheroma.

In short, my explanation is that the amount of regurgitation in these cases is so slight, and the stream of regurgitant blood passes the valves under such low pressure, that it is unable to determine an audible aortic murmur.

In the consideration of the facts disclosed in this paper the means employed for detecting sounds is of importance, I therefore mention that I use a binaural stethoscope and an ordinary wooden one, and that I am accustomed to listen to the heart sounds with both.

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